Invasive *Salmonella* Infections in the United States, FoodNet, 1996–1999: Incidence, Serotype Distribution, and Outcome

Duc J. Vugia,[1,2] Michael Samuel,[2] Monica M. Farley,[3] Ruthanne Marcus,[5] Beletshachew Shiferaw,[6] Sue Shallow,[2] Kirk Smith,[7] Frederick J. Angulo,[4] for the Emerging Infections Program FoodNet Working Group[a]

[1]California Department of Health Services and [2]California Emerging Infections Program, Berkeley; [3]Emory University School of Medicine and Atlanta Veterans Affairs Medical Center and [4]Centers for Disease Control and Prevention, Atlanta, Georgia; [5]Connecticut Emerging Infections Program, New Haven; [6]Oregon Health Division, Portland; and [7]Minnesota Department of Health, Minneapolis

Invasive *Salmonella* infections are severe and can be life threatening. We analyzed population-based data collected during 1996–1999 by the Foodborne Diseases Active Surveillance Network (FoodNet), to determine the incidences, infecting serotypes, and outcomes of invasive *Salmonella* infection. We found that the mean annual incidence of invasive salmonellosis was 0.9 cases/100,000 population and was highest among infants (7.8 cases/100,000). The incidence was higher among men than women (1.2 vs. 0.7 cases/100,000; $P < .001$) and higher among blacks, Asians, and Hispanics than among whites (2.5, 2.0, and 1.3 cases/100,000 population, respectively, vs. 0.4 cases/100,000; all $P < .001$). Seventy-four percent of cases were caused by 8 *Salmonella* serotypes: Typhimurium, Typhi, Enteritidis, Heidelberg, Dublin, Paratyphi A, Choleraesuis, and Schwarzengrund. Of 540 persons with invasive infection, 386 (71%) were hospitalized and 29 (5%) died; 13 (45%) of the deaths were among persons aged ≥60 years. Invasive *Salmonella* infections are a substantial health problem in the United States and contribute to hospitalizations and deaths.

Each year in the United States, *Salmonella* infects ~1.4 million persons, resulting in >16,000 hospitalizations and almost 600 deaths [1]. Although most *Salmonella* infections result in mild-to-moderate gastroenteritis that usually resolves with or without treatment, some lead to severe invasive infections (e.g., bacteremia and meningitis). From 1987 to 1997, 384,266 *Salmonella* isolates with a known clinical source were reported to the Centers for Disease Control and Prevention (CDC, Atlanta, GA), 23,237 (6%) of which were isolated from blood or CSF [2]. Invasive *Salmonella* infections can be life threatening and usually require hospitalization and treatment with parenteral antibiotics [3, 4]. Deaths from invasive *Salmonella* infections are not uncommon [3, 5].

Despite the severe nature of invasive salmonellosis, we found no published population-based determination of US incidences of invasive *Salmonella* infection. Although invasive *Salmonella* infections are known to occur more commonly among infants aged <1 year [2, 6, 7], elderly persons [6, 7], and immunocompromised persons [3, 4], including those who are infected with HIV [8–12], little has been published on invasive salmonellosis in the general population.

We analyzed population-based data collected during 1996–1999 by the Foodborne Diseases Active Surveillance Network (FoodNet), to determine the incidence of invasive *Salmonella* infections in the general population, to describe the demographic characteristics of those infected, to evaluate the *Salmonella* serotypes that

are most commonly associated with invasive disease, and to determine the outcomes for infected persons.

SUBJECTS AND METHODS

FoodNet, which was started in 1996, is a collaborative program among selected state health departments, the CDC, the US Department of Agriculture Food Safety and Inspection Service, and the US Food and Drug Administration [13, 14]. Although FoodNet has since expanded to include additional surveillance areas (also known as "FoodNet sites"), we analyzed data only from those FoodNet sites that performed surveillance during the entire 1996–1999 study period, to facilitate year-to-year comparison. The FoodNet study sites at that time included Minnesota, Oregon, and selected counties in California (Alameda and San Francisco), Connecticut (Hartford and New Haven), and Georgia (Clayton, Cobb, DeKalb, Douglas, Fulton, Gwinnett, Newton, and Rockdale). The postcensus estimates of the population within these areas were 14.3 million in 1996, 14.4 million in 1997, 14.6 million in 1998, and 14.8 million in 1999, representing approximately 5.4% of the population of the United States each year. FoodNet conducts population-based active surveillance for culture-confirmed cases of *Campylobacter, Escherichia coli* O157:H7, *Listeria, Salmonella, Shigella, Vibrio*, and *Yersinia enterocolitica* infections among the residents of these sites [14]. To ascertain culture-confirmed cases and to collect patient demographic information, including sex, age, and race/ethnicity, investigators contacted, at least once a month, clinical laboratories serving these residents. Information was collected on the source of the *Salmonella* isolate (e.g., stool, blood, and CSF) and whether the infected patient was hospitalized or died. Because clinical laboratories routinely submitted *Salmonella* isolates to their respective state public health laboratory for serotyping, serotype data were also collected. We conducted the study in accordance with guidelines for human research specified by the US Department of Health and Human Services.

At each site, data were routinely entered into a database and forwarded without personal identifiers to the CDC. We analyzed these data using SAS (SAS Institute) and Stata (Stata) software and made rate comparisons using the standard binomial method in Stata. We compared between-group differences in proportions using χ^2 or Fisher's exact tests and considered $P \leqslant .05$ to be significant. We calculated *Salmonella* incidences using appropriate 1996–1999 population estimates. We defined "invasive" cases as those in which *Salmonella* had been isolated from normally sterile sites–specifically from blood, CSF, peritoneal fluid, and bone or joint. We did not define isolation of *Salmonella* from urine or wounds as invasive because we lacked the necessary clinical data to distinguish between invasive infection and colonization or contamination

of urine and wounds. Invasive cases of *Salmonella* infection were analyzed by site; year; patients' sex, age group, and race/ethnicity; serotype; whether the patient was hospitalized; and whether the patient died. We defined "enteric" cases as those in which *Salmonella* had been isolated from stool specimens, including those collected by rectal swabs. We then compared culture-confirmed invasive cases with enteric cases, if appropriate. We submitted records of cases of *Salmonella* bacteremia in San Francisco County to the San Francisco Office of AIDS, to determine the proportion of patients with *Salmonella* infection who were also reported to have AIDS during 1996–1999.

RESULTS

During 1996–1999, there were 7895 culture-confirmed cases of *Salmonella* infection identified in the FoodNet study area. Of these, 7169 isolates (90.8%) were from stool or rectal swabs, 540 isolates (6.8%) were invasive infections, 93 isolates (1.2%) were from urine, 70 isolates (0.9%) were from other sources such as abscesses or wounds, and 23 isolates (0.3%) were of unknown origin. The proportion of culture-confirmed *Salmonella* infections that were invasive ranged from 11% in California to 3% in Minnesota. Of the 540 invasive isolates, 517 isolates (96%) were from blood, 16 isolates (3%) were from bone or joint, 3 isolates (0.6%) were from CSF, and 4 isolates (0.7%) were from peritoneal fluid.

The mean annual incidence of culture-confirmed invasive *Salmonella* infections for the FoodNet study area was 0.9 cases/100,000 population; the incidence ranged from 0.4 in Minnesota to 1.8 in California. When we stratified the California site by its 2 participating counties, we found a mean annual incidence of 2.4 cases/100,000 population in San Francisco County and 1.5 cases/100,000 population in Alameda County.

During 1996–1999, we found no clear trend in the percentage of culture-confirmed *Salmonella* infections that were invasive or in the overall mean annual incidence of invasive *Salmonella* infections. By site, there appeared to be a decreasing trend in annual incidences during the study period in California and in Connecticut, whereas the other 3 sites showed no clear trend (figure 1). The California site's annual incidence decreased from 2.0 cases/100,000 population in 1996 to 1.5 in 1999, with that of San Francisco County decreasing from 3.0 in 1996 to 1.6 in 1999. However, the decreases were not statistically significant for either California or Connecticut.

Culture-confirmed invasive *Salmonella* infections were more common among men than among women. The mean annual incidence was 1.2 cases/100,000 men, compared with 0.7 cases/100,000 women ($P < .001$); 62% of patients with invasive infection were male. For neither sex did the 1999 incidence differ significantly from that in 1996. Among male patients, the mean annual incidence was higher in San Francisco County (3.3 cases/
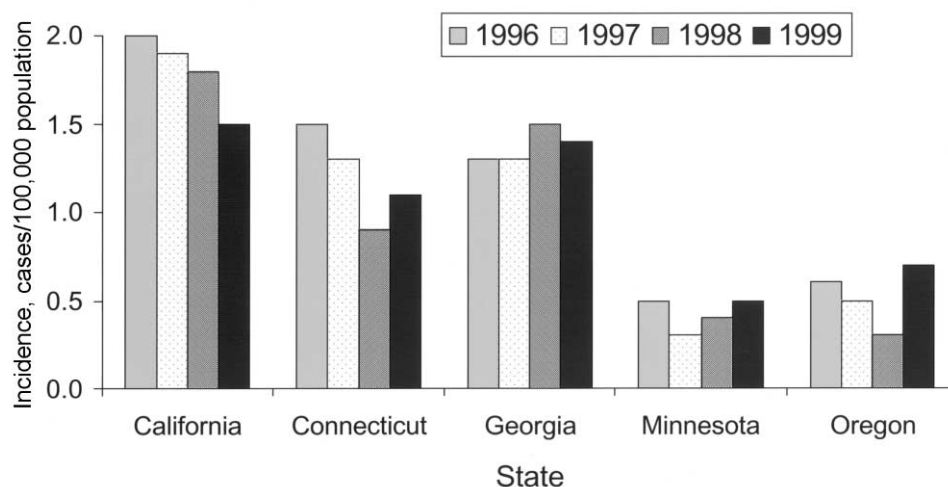
Figure 1. Incidence of invasive *Salmonella* infection, by FoodNet site and by year, 1996–1999

100,000 men) than in other FoodNet sites; San Francisco's annual incidence among male patients decreased from 4.1 cases/100,000 in 1996 to 2.2 cases/100,000 in 1999. Given the possible effect of male patients with HIV/AIDS on the incidence of invasive *Salmonella* infections in San Francisco, we also analyzed the overall incidence in FoodNet sites, by sex, while excluding patients from San Francisco County. In sites other than San Francisco County, the overall mean annual incidence was 1.1 cases/100,000 men, compared with 0.6 cases/100,000 women ($P < .001$).

By age group, the overall mean annual incidence peaked at 7.8 cases/100,000 infants (aged <1 year old), decreased to 1.6 cases/100,000 toddlers (aged 1–4 years), remained at ≤0.8 cases/100,000 older children and adults, then increased to 1.3 cases/100,000 persons aged ≥60 years. By age group and sex, the mean annual incidence for male patients was higher than that for female patients in all age groups except among children aged 5–9 years (figure 2); the incidence was highest among male infants aged <1 year (9.4 cases/100,000). When incidences by sex and age group were further evaluated by site, the mean annual incidence among male infants ranged from 3.4 cases/100,000 in Oregon to 14.5 cases/100,000 in Georgia, whereas, for female infants, it ranged from 0.8 cases/100,000 in Minnesota to 11.9 cases/100,000 in Georgia. Among persons aged ≥20 years, male patients in California had higher incidences than those at other sites (data not shown). When we stratified the California data by the state's 2 participating counties, we found that the mean annual incidences for male patients in San Francisco were higher than those for male patients in Alameda for all age groups. Among men in San Francisco in the 30–39, 40–49, and 50–59 age groups incidences were distinctly higher (4.0 cases/100,000 for all 3 age groups) than among these same groups of men in other FoodNet areas. Among San

Francisco men with culture-confirmed *Salmonella* infection, 18%, 21%, and 23% of those aged 30–39, 40–49, and 50–59 years, respectively, had invasive infections, compared with only 6.8% among all study patients with *Salmonella* infections.

The San Francisco Office of AIDS determined that, of San Francisco men aged ≥20 years who had *Salmonella* bacteremia during 1996–1999, there were 43% with a diagnosis of AIDS. The proportion with an AIDS diagnosis was even greater, 71%, among San Francisco men aged 35–49 years. Among those men aged ≥20 years, the proportion of those with *Salmonella* bacteremia who also had AIDS decreased from 60% in 1996 to 29% in 1999 (Suzan Scheer, San Francisco Office of AIDS, personal communication).

Data on race and ethnicity were available for 427 (79%) of patients with invasive infection. The mean annual incidences of culture-confirmed invasive *Salmonella* infections for blacks (2.5 cases/100,000), Asians (2.0 cases/100,000), and Hispanics (1.3 cases/100,000) were all higher than that for whites (0.4
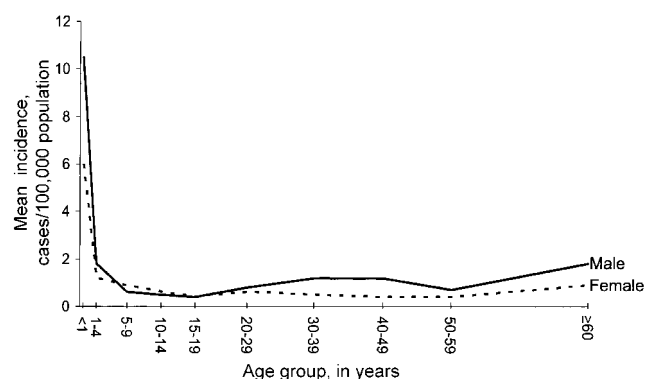
Figure 2. Mean annual incidence of invasive *Salmonella* infection, by sex and age group, original FoodNet sites, 1996–1999.

Table 1. Rates of *Salmonella* infection, by serotype, 1996–1999.

Serotype	No. of culture-confirmed cases	No. (%) of invasive cases	Percentage of invasive cases, by race/ethnicity of patients			
			White	Black	Hispanic	Asian
Typhimurium	2160	135 (6)	29	51	12	8
Typhi	106	76 (72)	18	12	14	57
Enteritidis	1094	71 (6)	43	36	17	3
Heidelberg	551	60 (11)	40	48	4	8
Dublin	28	20 (71)	71	18	0	6
Paratyphi A	27	17 (63)	13	0	13	75
Choleraesuis	23	12 (52)	11	0	22	67
Schwarzengrund	88	11 (13)	44	33	11	11
Other[a]	3818	138 (4)	…	…	…	…

[a] No other *Salmonella* serotype caused >9 invasive cases.

cases/100,000) (all $P < .001$). These racial and ethnic differences were similarly significant in each of the 5 FoodNet areas. Overall, by race and ethnicity, the proportion of cases of salmonellosis that were invasive was also higher among blacks (19%), Asians (15%), and Hispanics (9%) than among whites (5%) (all $P < .001$).

When we compared the incidences of invasive salmonellosis by racial/ethnic group for 1996 with corresponding rates for 1999, we found that the incidence for whites remained the same at 0.4 cases/100,000, whereas those for blacks, and decreased from 3.2 to 2.2 cases/100,000, for Asians from 2.4 to 1.9 cases/100,000, and for Hispanics from 1.8 to 1.0 cases/100,000, respectively. However, none of these decreases were significant. By age and race/ethnicity, black infants aged <1 year had the highest average annual incidence of 23.7 cases/100,000 population.

Seventy-four percent of 540 culture-confirmed invasive *Salmonella* infections were caused by 8 *Salmonella* serotypes (in descending order): Typhimurium (25%), Typhi (14%), Enteritidis (13%), Heidelberg (11%), Dublin (4%), Paratyphi A (3%), Choleraesuis (2%), and Schwarzengrund (2%) (table 1). For comparison, these 8 serotypes accounted for 50% of isolates from stool specimens. By serotype, the proportion of *Salmonella* infections that were invasive was relatively high for serotypes Dublin (80%), Typhi (75%), Paratyphi A (65%), and Choleraesuis (57%) and was much lower for serotypes Typhimurium (6%), Enteritidis (7%), Heidelberg (11%), and Schwarzengrund (14%).

When we analyzed these 8 serotypes by year and by site, the numbers were too small and variable to identify definite trends. The proportion of invasive infections among male patients was higher than that among female patients for all of these serotypes except for Typhi (data not shown). By age group, the numbers were small and were spread out among most age groups for most serotypes, except that no infections with the Dublin se-

rotype occurred among people aged <30 years and no infections with the Paratyphi A serotype occurred among children aged <5 years; for Choleraesuis, Dublin, and Enteritidis, the highest proportion of invasive cases were among those aged ≥60 years (50%, 50%, and 32%, respectively).

Invasive cases of *Salmonella* infection due to several serotypes occurred more frequently among Asians and blacks (table 1). The highest proportions of invasive Typhimurium and Heidelberg infections occurred among blacks, whereas the highest proportions of invasive Typhi, Paratyphi A, and Choleraesuis infection occurred among Asians. For comparison, although a disproportionate number of enteric Typhi (79%) and Choleraesuis (43%) infections occurred in Asians, enteric infections caused by the other 6 serotypes predominated in whites (data not shown).

Seventy-one percent (386 of 540) of patients with invasive *Salmonella* infection were hospitalized, compared with only 17% (1185 of 7169) of those with enteric infection ($P < .001$).
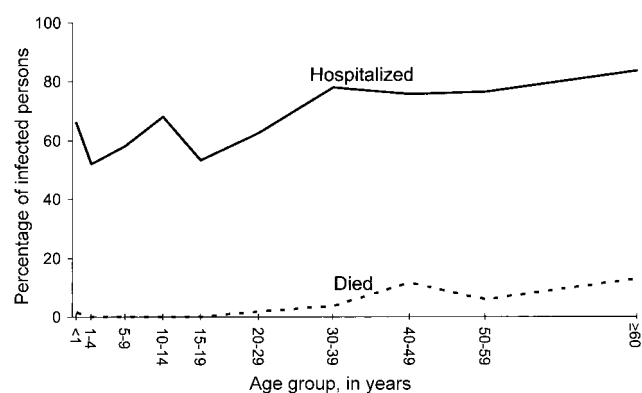
Figure 3. Percentage of persons with invasive *Salmonella* infection who were hospitalized and who died, by age group, original FoodNet sites, 1996–1999.

The proportion of patients with invasive infection who were hospitalized varied from 69%, in California and Georgia, to 82% in Connecticut. By year, the proportion of patients with invasive infections who were hospitalized decreased from 77% in 1996 to 69% in 1999 ($P = .08$; χ^2 test for trend). Hospitalization rates for female patients (75%) did not differ significantly from those for male patients (70%). However, the proportion of female patients with invasive salmonellosis who were hospitalized decreased significantly, from 84% in 1996 to 69% in 1999 ($P = .05$; χ^2 test for trend). The proportion of male patients who were hospitalized decreased from 75% in 1996 to 68% in 1999, but this was not significant ($P = .35$; χ^2 test for trend). By age group, the hospitalization rate among persons with invasive infection was highest among those aged ≥60 years (83%); among infants, 66% were hospitalized. There was a significant trend of increasing proportion of patients hospitalized with increasing age group ($P < 001$, χ^2 for trend; figure 3). The hospitalization rates among patients with invasive salmonellosis did not vary significantly by race/ethnicity: 76% among blacks, 75% among Asians, 70% among Hispanics, and 76% among whites.

Of 540 patients with invasive infection, 29 (5.4%) died, compared with 13 (0.2%) of 7169 patients with enteric infection ($P < .001$). Of the 29 decedents, 27 (93%) had *Salmonella* bacteremia and 2 had *Salmonella* isolated from peritoneal fluid. The proportion of patients with invasive infection who died did not change significantly over time; it was 9% in 1996 and 5% in 1999. The death rate among patients with invasive salmonellosis was 4% for male patients and 7% for female patients. By age group, the proportion of deaths was highest among persons aged 40–49 years. Of the total 29 deaths, 13 (45%) were of persons aged ≥60 years. There was a significant trend of increasing proportion of patients who died with increasing age group ($P < .001$; χ^2 test for trend; figure 3). By race and ethnicity, 8% of blacks, 6% of Asians, 2% of Hispanics, and 4% of whites with invasive infection died; these differences were not significant. By serotype, 18 (62%) of invasive infections that resulted in deaths were caused by *S*. Typhimurium, whereas 2 (7%) each were caused by *S*. Dublin and *S*. Newport.

DISCUSSION

During 1996–1999, the mean annual incidence of invasive *Salmonella* infection, primarily bacteremia, was 0.9 cases/100,000 persons in the FoodNet areas. Seventy-one percent of patients with invasive salmonellosis were hospitalized, and ∼5% died. The incidence of 0.9 was higher than the 0.3 rate reported for 1977 in the only previous US study of invasive *Salmonella* infection (bacteremia) [7].

Projecting the current incidence of invasive infection and the proportion of infected persons hospitalized and who died to the entire US population (using the 2000 census), we estimate that >2500 culture-confirmed cases of invasive salmonellosis occur annually in this country, which result in nearly 1800 hospitalizations and 150 deaths. The true totals, however, are likely higher, because not all invasive *Salmonella* infections are confirmed by culture. For example, some persons with invasive infection could have sought medical care and been treated empirically with antimicrobial agents before diagnostic specimens were considered or were obtained from them; such treatment would decrease the likelihood of isolating *Salmonella*. Mead et al. [1] considered this possibility when developing estimates of the burden of illness caused by foodborne diseases and adopted a correction factor of 1 undiagnosed case for each culture-confirmed case of serious infection. If we used Mead's correction factor, we would estimate that >5000 invasive *Salmonella* infections occur annually in the United States.

The higher population-based incidence of invasive salmonellosis among men has not been documented before. In reviewing all *Salmonella* isolates (most of which were from stool samples) reported to the CDC between 1987 and 1997, Olsen et al. [2] noted that, among children aged <15 years, boys were slightly more likely to be infected, but that, among adults aged 20–74 years, the infection rate was 12.2 cases/100,000 women versus 8.8 cases/100,000 men. However, Olsen et al. [2] did not stratify isolation rates for invasive salmonellosis by sex. In the earlier CDC report of cases of *Salmonella* bacteremia during 1968–1979, it was reported that 56% of blood isolates and 51% of stool isolates were from men [7]. Results of a review of septicemic nontyphoidal salmonellosis in New York City during 1962–1971 indicated that 59% of those infected were male [6].

What would account for a difference in rates of invasive salmonellosis by sex? Although the increased risk for *Salmonella* bacteremia among persons with HIV/AIDS may partially explain why rates of invasive cases among people aged 30–50 years were higher among men than women, the overall higher rates among male patients, particularly among infants, children aged <5 years, and those aged ≥60 years, cannot be fully explained. Even when the San Francisco data (which included men with HIV/AIDS) were excluded, rates among men were still significantly higher than rates samong women. This male predominance among patients with invasive infections was present for each of the most common 8 *Salmonella* serotypes except for Typhi. Of interest, infection with *Campylobacter*, another foodborne pathogen, also has a higher incidence among males, beginning in infancy and persisting through older age groups [15]. Among possible reasons for this difference in incidences by sex are host factors, such as a postulated higher male susceptibility to certain infectious diseases [16], and behavioral factors, such as the reported tendency of males to engage in more high-risk food handling, preparation, and consumption behaviors [17].

We found blacks, Asians, and Hispanics to have higher population-based incidences of invasive *Salmonella* infections than whites, and this too has not been documented previously. The reasons for these higher incidences may involve host factors and exposures particular to these populations, including foreign travel and eating certain ethnic foods. For example, Mermin et al. [18] reviewed cases of typhoid fever in the United States between 1985 and 1994 and found that 72% of those infected had traveled internationally within 30 days before the onset of illness, primarily to Mexico, India, the Philippines, Pakistan, El Salvador, and Haiti. They found that the overall rate of typhoid fever among travelers who were not US citizens (and therefore more likely to be nonwhite visitors to foreign homelands) was greater than among those who were citizens. More recently, outbreaks of *S.* Typhimurium DT104 were reported among Hispanics in California and Washington who ate homemade Mexican-style soft cheeses made from raw milk [19, 20]. Also, the high proportion of infection with swine-adapted *S.* Choleraesuis found among Asians may possibly be explained by the popularity of pork in Asian communities. Further research for risk or host factors for *Salmonella* infection within these diverse populations may help to explain their higher incidences of invasive disease.

The rates of hospitalization and death among people with invasive *Salmonella* infection were highest among elderly persons. Possible reasons for this include senescence of gut immunity, the impaired function of macrophages and granulocytes, and other host factors, such as concurrent chronic underlying illnesses [21–24].

Although rates of hospitalization and death among those with invasive salmonellosis were highest among elderly persons, the incidence of invasive salmonellosis was highest among infants aged <1 year. And although the high incidence of invasive salmonellosis among infants has been well documented for decades [6, 7], the reasons for such consistently high rates in this age group are still unclear. Some researchers have postulated that gastric hypoacidity or gut immaturity may make infants more susceptible [3, 23]. The few researchers who looked at risk factors for *Salmonella* infection in this age group found the home environment and intrafamiliar transmission to be important risk factors but found breastfeeding to be protective [25–27].

Decreased host immunity caused by other conditions such as cancer or HIV infection has also been shown to contribute to the risk for *Salmonella* bacteremia [3, 4, 11, 21]. We did not collect information on underlying health conditions and therefore cannot speculate on what proportion of these cases may have been due to host immunosuppression. In San Francisco County, however, we did confirm with the San Francisco AIDS Office that a high proportion of adult men with *Salmonella* bacteremia during the study period were also included in the AIDS Registry. We also found that, during 1996–1999, there were decreases in both the annual incidence of invasive salmonellosis among male patients in San Francisco and the proportion of adult San Francisco men with *Salmonella* bacteremia who also had AIDS—decreases that coincided with the observed decrease in the incidence of AIDS and other opportunistic infections among persons with HIV/AIDS [28, 29]. The widespread use of HAART among persons with HIV/AIDS during these years was probably an important factor in these decreases [30]. The decrease in the number of cases of invasive salmonellosis in this immunocompromised population in San Francisco contributed disproportionately to the decrease in the incidence of invasive salmonellosis for the California FoodNet site during these years.

Seventy-four percent of invasive *Salmonella* infections were caused by only 8 *Salmonella* serotypes, but it has been shown previously that only a few *Salmonella* serotypes are responsible for most cases of invasive salmonellosis [2, 7, 12]. Invasiveness is thought to be facilitated by virulence plasmids and/or by chromosomal genes that express invasive factors [31, 32]. In our study, serotypes Typhi, Paratyphi A, Dublin, and Choleraesuis seem to have been highly invasive, because they made up a high serotype-specific proportion of invasive culture-confirmed infections; the invasiveness of these serotypes has been noted elsewhere [7]. Although serotypes Typhimurium, Enteritidis, and Heidelberg made up a low proportion of serotype-specific invasive infections, they accounted for substantial numbers of invasive cases, because they caused the most enteric infections. Why Asians and blacks had higher rates of invasive salmonellosis caused by certain serotypes is unknown, and the cause of these higher rates merits further research.

Our study had several limitations. Although active surveillance identified all cases of *Salmonella* infection diagnosed by culture, we would have missed any cases of invasive *Salmonella* infection for which a culture was not performed. As mentioned above, we did not uniformly collect data on patients' HIV status and other underlying conditions; therefore, we could not identify host factors associated with invasive *Salmonella* infection. Finally, despite the large population covered under FoodNet, our findings may not be representative of the general US population.

Although we did not obtain details on treatment, including antibiotic therapy received by patients with invasive *Salmonella* infection, the 71% hospitalization rate and the 5% mortality rate among these patients are cause for concern and demonstrate the seriousness of such infections. Further increases in the antimicrobial resistance of *Salmonella* [33, 34] may cause these rates to rise even further.

Invasive *Salmonella* infections are a substantial health problem in the United States and contribute to hospitalizations and deaths, particularly among elderly persons. Infants aged <1 year

have the highest incidence. Of note, the incidence is higher among men than among women, and higher among blacks, Asians, and Hispanics than among whites. Further studies are needed to determine why invasive *Salmonella* infections are more common in these populations and why certain *Salmonella* serotypes are disproportionately represented among the infections in certain groups. Such studies may lead to better efforts to prevent invasive salmonellosis and to decrease the resultant morbidity and mortality in affected populations.

FOODNET WORKING GROUP MEMBERS

CDC: Frederick Angulo, Timothy Barrett, Michael Beach, Nancy Bean, Richard Bishop, Thomas Boyce, Laura Conn, Vance Dietz, Mary Evans, Cindy Friedman, Kate Glynn, Patricia Griffin, John Hatmaker, Peggy Hayes, Debra Helfrick, Thomas Hennessy, Mike Hoekstra, Lori Hutwagner, Beth Imhoff, Malinda Kennedy, Deborah Levy, Bill MacKenzie, Kathleen Maloney, Nina Marano, Paul Mead, Thomas Navin, Sarah Pichette, Robert Pinner, Vasudha Reddy, Laurence Slutsker, Karen Stamey, Bala Swaminathan, David Swerdlow, Robert Tauxe, Thomas Van Gilder, Drew Voetsch, David Wallace, Stephanie Wong, and Samantha Yang Rowe. California: Sharon Abbott, Felicia Chi, Pam Daily, Marianne David, Mary Ann Davis, Lisa Gelling, Alexander McNees, Janet Mohle-Boetani, Nandeeni Mukerjee, Joelle Nadle, Jan O'Connell, Judy Rees, Kevin Reilly, Art Reingold, Gretchen Rothrock, Michael Samuel, Sue Shallow, Ben Silk, Duc Vugia, and Ben Werner. Connecticut: Gary Budnick, Matthew Cartter, Terry Fiorentino, James Hadler, Robert Howard, Gazala Kazi, Aristea Kinney, Ruthanne Marcus, Donald Mayo, Patricia Mshar, Randall Nelson, Quyen Phan, Robin Ryder, and Charles Welles. Georgia: Sabrina Burden, Molly Bardsley, Wendy Baughman, Paul Blake, Shama Desai, Monica Farley, Katherine Gibbs-McCombs, Laura Gilbert, Jane Koehler, Mina Pattani, Susan Ray, Matthew Sattah, Suzanne Segler, Kathleen Toomey, and Sabrina Whitfield. Maryland: Bernadette Albanese, Lillian Billman, Alicia Bustamante, Amy Carnahan, Michael Carter, Marcia Criscio, Yvonne Deane-Hibbert, Diane Dwyer, Lora Gay, Althea Glenn, Charmaine Gregg, Lee Harrison, Kelly Henning, Yvonne Hibbert, Kim Holmes, Jackie Hunter, Judith Johnson, Tobi Karchmer, Melissa Kent, J. Glenn Morris Jr., Lola Olabode, Peggy Pass, Jafar Razeq, Jeffery Roche, Dale Rohn, Christine St. Ours, Christian Steiner, Alexander Sulakvelidze, Frances Yarber, and Yongyu Wang. Minnesota: Jeff Bender, John Besser, Richard Danila, Valerie Deneen, Craig Hedberg, Julie Hogan, Heidi Kassenborg, Carlota Medus, Michael Osterholm, Kirk Smith, Dana Soderlund, and Julie Wicklund. New York: Bridget Anderson, Dianna Bopp, Hwa-Gan Chang, Kathy Carlton, Barbara Damaske, Nellie Dumas, Marie Fitzgerald, Karim Hechemy, Jonathan Hibbs, Julia Kiehlbauch, Dale Morse, Candace Noonan, Brian Sauders, Perry Smith, Nancy Spina, Cathy Stone, and Shelley Zansky. Oregon: Vijay Balan, Chris Biggs, Maureen Cassidy, Paul Cieslak, Emilio DeBess, David Fleming, Bill Keene, Stephen Ladd-Wilson, Lore Lee, Eileen Lorber, Steve Mauvais, Teresa McGivern, Beletshachew Shiferaw, Bob Sokolow, Regina Stanton, and John Townes. Tennessee: Brenda Barnes, Effie Boothe, Allen Craig, Diane Eigsti Gerber, Timothy Jones, William Moore, William Schaffner, and Pat Turri. US Department of Agriculture, Food Safety and Inspection Service: Arthur Baker, Ruth Etzel, Jill Hollingsworth, Peggy Nunnery, Phyllis Sparling, and Kaye Wachsmuth. US Food and Drug Administration, Center for Food Safety and Applied Nutrition: Sean Alterkruse, Ken Falci, Bing Garthright, Janice Oliver, and Clifford Purdy.

References

1. Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. Emerg Infect Dis **1999**; 5:607–25.
2. Olsen SJ, Bishop R, Brenner FW, et al. The changing epidemiology of *Salmonella:* trends in serotypes isolated from humans in the United States, 1987–1997. J Infect Dis **2001**; 183:753–61.
3. Hohmann EL. Nontyphoidal salmonellosis. Clin Infect Dis **2001**; 32: 263–9.
4. Miller SI, Pegues DA. *Salmonella* species, including *Salmonella typhi*. In: Mandell GL, Bennett JE, Dolin R, eds. Mandell, Douglas, and Bennett's principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, **2000**.
5. Villar R, Bardsley M, Reddy S, et al. Deaths from enteric bacterial pathogens: a chart review of deaths occurring in the Foodborne Diseases Active Surveillance Network (FoodNet) in 1996 [abstract P-3.14]. In: Program and abstracts of the International Conference on Emerging Infectious Diseases (Atlanta). Atlanta: Centers for Diseases Control and Prevention, **1998**:84–5.
6. Cherubin CE, Neu HC, Imperato PJ, Harvey RP, Bellen N. Septicemia with non-typhoid *Salmonella*. Medicine (Baltimore) **1974**; 53:365–76.
7. Blaser MJ, Feldman RA. Salmonella bacteremia: reports to the Centers for Disease Control, 1968–1979. J Infect Dis **1981**; 143:743–6.
8. Sperber SJ, Schleupner CJ. Salmonellosis during infection with human immunodeficiency virus. Rev Infect Dis **1987**; 9:925–34.
9. Celum CL, Chaisson RE, Rutherford GW, Barnhart JL, Echenberg DF. Incidence of salmonellosis in patients with AIDS. J Infect Dis **1987**; 156:998–1002.
10. Gruenewald R, Blum S, Chan J. Relationship between human immunodeficiency virus infection and salmonellosis in 20- to 59-year-old residents of New York City. Clin Infect Dis **1994**; 18:358–63.
11. Angulo FJ, Swerdlow DL. Bacterial enteric infections in persons infected with human immunodeficiency virus. Clin Infect Dis **1995**; 21(Suppl 1):S84–93.
12. Brown M, Eykyn SJ. Non-typhoidal *Salmonella* bacteraemia without gastroenteritis: a marker of underlying immunosuppression: review of cases at St. Thomas' Hospital, 1970–1999. J Infect **2000**; 41:256–9.
13. Angulo FJ, Voetsch AC, Vugia D, et al. Determining the burden of

human illness from foodborne diseases. In: Tollefson L, ed. The veterinary clinics of North America: food animal practice; microbial foodborne pathogens, March 1998. Philadelphia: WB Saunders, **1998**: 165–72.

14. Centers for Disease Control and Prevention. Incidence of foodborne illnesses: preliminary data from the Foodborne Diseases Active Surveillance Network (FoodNet)—United States, 1998. MMWR Morb Mortal Wkly Rep **1999**; 48:189–94.

15. Samuel M, Vugia D, Shallow S, et al. Epidemiology of sporadic *Campylobacter* infection in the United States and declining trend in incidence, FoodNet 1996–1999. Clin Infect Dis **2004**; 38(Suppl 3):S165–74 (in this issue).

16. Green MS. The male predominance in the incidence of infectious diseases in children: a postulated explanation for disparities in the literature. Int J Epidemiol **1992**; 21:381–6.

17. Yang S, Leff MG, McTague, et al. Multistate surveillance for food-handling, preparation, and consumption behaviors associated with foodborne diseases: 1995 and 1996 BRFSS food-safety questions. MMWR Surveill Summ **1998**; 47(SS-4):33–57.

18. Mermin JH, Townes JM, Gerber M, Dolan N, Mintz ED, Tauxe RV. Typhoid fever in the United States, 1985–1994. Arch Intern Med **1998**; 158:633–8.

19. Cody SH, Abbott SL, Marfin AA, et al. Two outbreaks of multidrug-resistant *Salmonella* serotype Typhimurium DT104 infections linked to raw-milk cheese in Northern California. JAMA **1999**; 281:1805–10.

20. Villar RG, Macek MD, Simons S, et al. Investigation of multidrug-resistant *Salmonella* serotype Typhimurium DT104 infections linked to raw-milk cheese in Washington State. JAMA **1999**; 281:1811–6.

21. Morris JG Jr, Potter M. Emergence of new pathogens as a function of changes in host susceptibility. Emerg Infect Dis **1997**; 3:435–41.

22. Khanna KV, Markham RB. A perspective on cellular immunity in the elderly. Clin Infect Dis **1999**; 28:710–3.

23. Shimoni Z, Pitlik S, Leibovici L, et al. Nontyphoid *Salmonella* bacteremia: age-related differences in clinical presentation, bacteriology, and outcome. Clin Infect Dis **1999**; 28:822–7.

24. Smith JL. Foodborne illness in the elderly. J Food Prot **1998**; 61: 1229–39.

25. Schutze GE, Sikes JD, Stefanova RCMD. The home environment and salmonellosis in children. Pediatrics **1999**; 103:E1.

26. Wilson R, Feldman RA, Davis J, LaVenture M. Salmonellosis in infants: the importance of intrafamilial transmission. Pediatrics **1982**; 69:436–8.

27. Yang S, Rocourt J, Shiferaw B, et al. Breast feeding decreases risk of salmonellosis among infants in FoodNet sites. In: Program and abstracts of the 36th Annual Meeting of the Infectious Disease Society of America (Denver). Alexandria, VA: Infectious Diseases Society of America, **1998**.

28. Nuorti PJ, Butler JC, Gelling L, Kool JL, Reingold AL, Vugia DJ. Epidemiologic relation between HIV and invasive pneumococcal disease in San Francisco County, California. Ann Intern Med **2000**; 132:182–90.

29. Palella FJ Jr, Delaney KM, Moorman AC, et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. N Engl J Med **1998**; 338:853–60.

30. Vittinghoff E, Scheer S, O'Malley P, Colfax G, Holmberg SD, Buchbinder SP. Combination antiretroviral therapy and recent declines in AIDS incidence and mortality. J Infect Dis **1999**; 179:717–20.

31. Guiney DG, Fang FC, Krause M, Libby S, Buchmeier NA, Fierer J. Biology and clinical significance of virulence plasmids in *Salmonella* serovars. Clin Infect Dis **1995**; 21(Suppl 2):S146-51.

32. Janda JM, Abbott SL. The salmonellae. In: The enterobacteria. Philadelphia, PA: Lippincott-Raven **1998**:80–100.

33. Lee LA, Puhr ND, Maloney K, Bean NH, Tauxe RV. Increase in antimicrobial-resistant *Salmonella* infections in the United States, 1989–1990. J Infect Dis **1994**; 170:128–34.

34. Glynn MK, Bopp C, Dewitt W, et al. Emergence of multidrug-resistant *Salmonella* enteria serotype Typhimurium DT104 infections in the United States. N Engl J Med **1998**; 338:1333–7.